HISPIDUS CANKERS OF HARDWOODS

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The fungus, Inonotus hispidus (Bulliard ex Fr.) Karsten (Polyporus hispidus Bull, ex Fr.), causes "heartrot" in a variety of living hardwood trees (1,2,4,6,7,10,11,12). This decay is usually accompanied by characteristic cankers along the stems of infected trees resulting from the fungus' ability to invade and kill living sapwood and cambial tissues as well (2,4,7,11,12). This capacity to cause both heartrot and coincident cankers on living trees places I. hispidus in a rather select group of fungi often referred to as canker-rot fungi (2,7). Notwithstanding, "hispidus canker" has become the generally accepted common name for the disease.

DISTRIBUTION, HOSTS, & DAMAGE. Hispidus cankers are common on hardwood tree species throughout much of the United States (1,2,4,6,7,10,11,12). Overholtz (10) lists Carya, Fraxinus, Juglans, Morus, Quercus, Salix, and Schinus as host genera for I. hispidus and notes that on rare occasions the fungus may occur on conifers in the genera Abies and Pinus as well. Oaks in the so-called red or black oak group (Erythrobalanus) are particularly susceptible (2,12). Graves (4) reported the occurrence of the pathogen on Fraxinus excelsior L. in England and cites Prillieux and Butler (literature inaccessible for this writing), respectively, as reporting the fungus causing damage to mulberry in France and mulberry as well as apple, plum, and apricot in India. In Florida, the author has observed hispidus cankers on laurel (Quercus laurifolia Michx.), southern red (Q. falcata Michx.), and turkey (Q. laevis Walt.) oaks.

Although not considered a major pathogen in the United States (from an overall impact standpoint), I. hispidus can cause significant damage to individual trees and, on occasion, to sizeable Stands of hardwood timber as well. Limited surveys in Connecticut (11) and Mississippi (12) revealed thai, in some areas up to 13% of the individual trees in certain oak species were infected with the pathogen. Wood volume losses due to the decay associated with hispidus cankers have been estimated to average between 2 and 7% (11,12), although losses of about 33% were estimated on one 1/10 acre site (11). Additional and potentially more serious losses may be realized in terms of stem breakage and subsequent mortality resulting from the structurally debilitating effects of the pathogen-induced heartrot (1,4).

PATHOGEN BIOLOGY & DISEASE DEVELOPMENT. Infection occurs primarily through dead branch stubs on the stems of susceptible hosts by means of aerially disseminated basidiospores (7,9,11,12). In many cases these stubs occur naturally as a result of the normal "self pruning" of lower branches. (Although not demonstrated, it is reasonable to assume that infections may also occur through other "entry courts" such as broken branches and/or deep wounds which expose susceptible heartwood tissues.) The fungus grows into the heartwood where it becomes well established, causing a spongy, straw-colored to pale yellow-brown delignifying rot, before growing outward through the living sapwood into the cambial tissues (2,4,7,11,12). As the pathogen invades and kills the cambium, conspicuous callus "folds" develop around canker margins giving the cankers a characteristic elongate, swollen appearance (Fig. 1). Bark on the canker "faces" remains firmly attached and appears sunken due to the cessation of radial (cambial) growth in cankered tissues. Branch stubs or scars are common, although not necessarily detectable, at or near the center of canker faces.





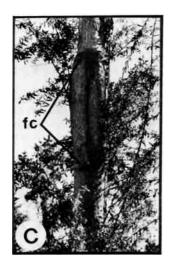


Fig. 1. Hispidus cankers on oaks in Florida. Note conspicuous callus folds at canker margins (A,B), spindle-shaped swelling of infected stem (C), and characteristic fungus conks (fc) attached to canker faces. Fresh conks are light colored while old conks and/or conk remnants are dark.

Hispidus cankers, and presumably the concomitant heartrot, develop vertically (longitudinally) at an estimated rate of ca. 6" per year (12). Radial (horizontal) growth of the fungus occurs at a much slower rate (11). Being perennial in habit (i.e., active for several years), hispidus cankers can, and often do, reach rather impressive ages. Sleeth and Bidwell (11), for example, reported 30-year-old cankers on oaks in Connecticut. "Average" hispidus cankers are approximately 3-4' long, but lengths of up to 14' have been reported (11). As a rule, heartrot resulting from I. hispidus infections can be expected to extend ca. 12-15" above and below the readily identifiable extremities of typical hispidus cankers (11,12).

During the late summer, fall, and early winter (7,8,9,12), I. hispidus produces large, spongy, bracket-shaped conks (sporophores) on the canker faces (Fig. 1). These conks are yellowish brown to rusty red or brown in color, and somewhat watery when fresh. A dense hirsute or "hispid" tomentum (i.e., dense, somewhat stiff, tufted or matted hairs) covers the upper surface (pileus) of the conks. The lower, spore-bearing surface (hymenium) is poroid. With time, the conks darken to nearly black, become hard, and often fall from the tree. Remnants of old I. hispidus conks are often detectable months later, either still attached to canker faces or lying on the ground at the bases of infected trees.

CONTROL. No effective therapeutic treatment is known for trees infected with I. hispidus. Control strategies in both forest and urban settings must be based on the concepts of prevention, sanitation, and salvage. Some guidelines follow.

Timber Stands

Harvest and salvage infected stems where practicable. In timber stand improvement (TSI) operations, fell infected trees to minimize sporophore production and spore dispersal (5,7,9). Removal or felling of infected trees provides the additional benefits of reducing competition for the more desirable, disease-free trees and reducing the risk of injury to healthy trees through stem breakage and falling of cankered trees. Avoid unnecessary logging or other injury to stems of susceptible host species.

Landscape or Urban Settings

Identify and remove trees with hispidus cankers as "hazard trees" with respect to stem breakage and inoculum reservoirs (i.e., sources of infective spores). Time pruning of host tree species so as to minimize exposure of susceptible tissues: a) prune stem branches when sufficiently small to facilitate the "healing" process of callus formation and b) perform such pruning in late winter or spring when spores of the pathogen are not being disseminated. Avoid unnecessary injuries to stems of susceptible trees.

SURVEY AND DETECTION. Look for elongate, swollen cankers displaying conspicuous callus folds on the main stems of susceptible hardwood species. Canker faces will appear characteristically sunken. Large, spongy, yellowish brown to rusty brown, bracket-shaped conks may appear on canker faces in late summer to early winter. At other times of the year, look for hard, darkened remnants of conks on canker faces or the ground beneath.

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